A TOUR OF THE SOUTH-WESTERN PACIFIC

BY

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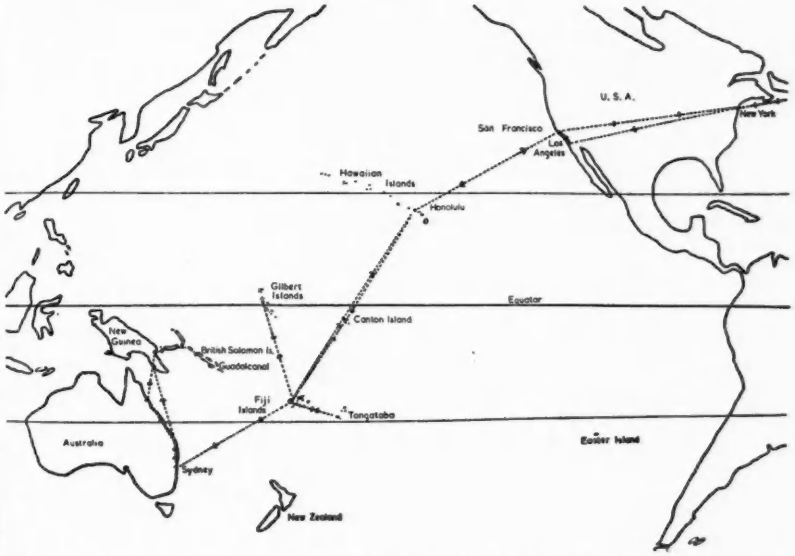
I returned from a six-day holiday in the Scilly Isles to be greeted by an invitation from the Colonial Office to visit the South Pacific Islands as a medical visitor. What an opportunity! An immediate acceptance seemed indicated and fortunately the United Bristol Hospitals and the South Western Regional Hospital Board agreed to my being seconded; also my colleagues very kindly accepted the extra work involved. Six weeks later I was away on a ten weeks' trip abroad, as rich in experience as any that I have had the good fortune to enjoy.



Map of the Pacific Ocean to show the route travelled and the places visited. The tour was centred on the Fiji Islands.

I was to be a medical visitor and the letter explained the two-fold purpose of my visit; to advise and assist in any medical matter in which I could help, not only in orthopaedic surgery, and to meet as many medical men in these islands as was possible, in order to break down the sense of isolation which easily enfolds those working so far from the centres of population. I had heard of these medical visitors some eight years earlier and had agreed to co-operate when I was asked; the scheme then only embraced Africa. Time passed and we had decided at home that, as far as I was concerned, what I had foreseen had been but a mirage. Now the reality, and the South Sea Islands instead of Darkest Africa. The Director of Medical Services, South-Western Pacific, had requested that the first medical visitor be an orthopaedic surgeon, and in due course the invitation had found its way into my letter-box.

The territory to be visited comprised the Fiji Islands, the Gilbert and Ellice Islands and the British Soloman Islands (see map). New Hebrides was originally included but had to be ruled out as no transport was available, but a short day visit to Tonga was added later.

Travelling was by air throughout the trip, the double-decker Boeing Stratocruiser, the DC7C, the Superconstellation, the Sunderland Flying Boat as the guest of the New Zealand Air Force, the Dakota and the little Rover of the Fiji "Scareways". I had originally been primed for "a rather long journey in a rather small boat" which I later found to be planned to take me from the Gilbert Islands to the British Soloman Islands. Fortunately, the High Commissioner was using the boat and I was returned to Fiji to reach Guadalcanal in the Soloman Islands by travelling to Sydney, Brisbane, Port Moresby and Lae, and spending the last day of the three days' journey hopping from island to island in a Dakota aircraft, passing through Rabaul and reaching Honiara, the capital, just before nightfall.

On retracing my steps to Port Moresby, I stayed there for three days at the request of the Australian Administration, and saw their tuberculous spines and leprosy cases. Medical visitors are comparatively rare on that route and one gets one's specialist advice when opportunity offers. I sometimes represent this trip as a giant "Domiciliary Visit", but it was a long time since the last such visit to some places.

I was very fortunate in being well cared for by many new-found friends, and I usually stayed in their homes and was passed from one to another as I moved about the territory.

Bristol was well represented. Dr. C. Gurd is the Medical Specialist at Suva and the only Medical Specialist in the British South Western Pacific territories; Dr. Downes is the Medical Officer to Nandi Airport; Dr. Bauman in Tonga had been in general practice in Bristol, and two other medical men had worked as house surgeons in Bristol during the last ten years.

The medical service varied from one territory to another, being most advanced in Fiji which, having a European population of over 9,000 and being on the main air route between San Francisco and Australia and New Zealand, is no backwater in this contracting world. Fundamentally, the services are similar in that most people primarily consult a medical man with less training and experience than that provided for in the qualifying medical examinations of most countries. The Assistant Medical Practitioner, trained in Fiji with a course now extending over five years, provides the basic practitioner of medicine in most of the South Pacific Islands. The Central Medical School at Suva, the capital of Fiji, takes students from Samoa, Tonga, the Gilbert and Ellice Islands, the British Solomon Isles and from New Guinea. The school has about 120 students and qualified its first women students last year. In all the Islands, the Assistant Medical Practitioner practises medicine in the villages and in the hospital, and is supervised in both by the Medical Officers, or in the occasional large hospital by the Medical and Surgical Specialists. In some territories where the islands are even more scattered, as for instance in the Solomon Islands and the Gilbert and Ellice Islands, resort has to be had to Dressers—medical aides with far less knowledge and experience, but who comprise the only reasonable unit to be scattered so widely to serve so small a population. In New Guinea, the medical services have been developed along different lines, using Europeans with a modicum of medical knowledge recruited from those who served in the medical services of the three Forces or with previous hospital or first aid experience. These European Medical Assistants have supplied the basic medical services for the scattered population.

The Medical Officer always supervises the services of a wide area, controlling the work in the large hospitals and the very important Public Health and Preventive Medicine aspects of the whole area. New Guinea is now training many Assistant Medical Practitioners in the Central Medical School at Suva to develop a basic medical service from the many races native to New Guinea.

While in Suva, I took the students on surgical rounds on several occasions and

found some quite promising and intelligent students amongst them. At the moment, it is not possible to make any advance from the Assistant Medical Practitioner qualification; it is necessary to start again in a University in New Zealand or Australia. The World Health Organization has taken a great interest in the Central Medical School, Suva, and has provided two extra lecturers in the pre-clinical subjects. There is a strong feeling that progress should be made towards getting the pre-clinical subjects up to a standard acceptable to some university, so allowing a promising Assistant Medical Practitioner later to take a full medical qualification by further study in the clinical subjects alone.

One very promising Assistant Medical Practitioner, practising surgery in one island I visited, was granted an "honorary matriculation" for his services to the Army of his country during the war. I was interested to find that he was thereby enabled to study at Dunedin; the first year he passed his biology, the second his chemistry and the third year he returned to his surgery in his home town. Basic education, and particularly weakness in mathematics, were the reason for the failure of this wholly laudable, if unconventional, academic exercise.

The Central Medical School, Suva, is staffed by some excellent teachers with a great sense of vocation, and it provides a fundamental service to medicine in these islands. The training of Assistant Medical Practitioners is not a temporary expedient, but will continue to provide the basic practitioner in medicine to these myriad of islands, where geography compels the community to live in units too small to provide either the remuneration or the clinical interest sufficient to hold a man or woman more highly qualified.

When I operated in Suva, the anaesthetics were always given by an Assistant Medical Practitioner, and the very pleasant and capable surgeon in Nukualofa, the capital of Tonga, is an Assistant Medical Practitioner. There are many such able practitioners of medicine and surgery among their ranks, but a full medical qualification must demand a basic education which the older generation of Assistant Medical Practitioners has not had the good fortune to enjoy.

The Medical Officer I met was usually quite young and enthusiastic, but practising quite a different brand of medicine than his contemporary at home. Public Health and Preventive Medicine affecting the villages, labour lines, scattered houses and the townships take much of his time. Supervision of the Assistant Medical Practitioners in his area involves much travelling, often on horseback or by boat, and control of his hospital with such surgery as he deems advisable, due allowance being given to the nursing in these smaller units as well as to his own ability, adds further variety. To the European population he often acts as general practitioner, although in many places the primary service is given by the Assistant Medical Practitioner, and geography might well result in his being the sole bastion between his friend and some fell disease which, in England, would be looked upon as almost the exclusive province of some less well-known speciality. I met Medical Officers wholly responsible for almost all the medicine, surgery, obstetrics and all the specialities, not forgetting dental surgery and even an occasional veterinary case: save in the last, his patients include a European section. Evacuation is difficult and erratic and only occasionally enables him to obtain the help of other medical men. The various territories largely deal with their own problems, but the medical and surgical specialists at Suva make it a focal point for consultations or surgery, while other patients go further afield to Australia and New Zealand, from whence are derived many of the white people living in these islands.

The nursing staff comprises European sisters recruited from Australia and New Zealand or the United Kingdom, Fiji-trained nurses from the Suva school of nursing and untrained assistants.

The Sisters of St. Mary provide a wonderful service for the lepers at Makogai in the Fiji group and in the Leper Colony on Guadalcanal. Their cheerful efficient service, so humanely rendered to these unfortunates, has been dramatically rewarded

since 1948 by the amazing results which have resulted from the introduction of the sulphone group of drugs.

The disease pattern is very different from our own. Appendicitis and peptic ulcers are exceedingly rare, trauma is not very prominent and, in its severer form in Fiji, is often the result of an attack by an Indian on an Indian; malignant disease is seen in smaller numbers than in Europe, but the population has a lower average age. Undernutrition and anaemia are usually considered to be among the more common ailments suffered by the Indian population in Fiji, more so among the women with their large families. The Fijians are usually much fitter but are very prone to tuberculosis, which is the major problem in these islands. It is difficult to assess the relative incidence of tuberculosis in Fiji between the two races as the Indians, under social and economic stress, will not stay in the chest hospital, while the Fijians, with the security of the village life for all their dependants, occupy the hospital beds, content to accept the treatment and fill the evening air with their delightful music and song. In Fiji, mass radiography and B.C.G. innoculation for children all feature in a vigorous programme designed to control this most serious of their diseases.

In the countries I visited, malaria was absent from Fiji and the Gilbert Islands, but was a very important cause of ill health in New Guinea and the Solomon Islands.

Filariasis is not infrequent, causing lymphangitis and giving positive blood smears, but elephantiasis is quite rare, save among the Ellice Islanders; this late result appears to follow very many years of infection.

Yaws in all the territories is reeling beneath the blow of 1 Mega unit of penicillin to every head of population, prescribed by the World Health Organization and delivered by teams of local Assistant Medical Practitioners under its direction. Is this single blow fatal? We have yet to find the answer to this question, but the lesions are becoming very rare and penicillin has remarkable curative properties in this disease.

Tetanus appears to be much more common than in England, and I asked for an injection of A.T.S. when I came off my Vespa just in case I might prove unlucky. The results of treatment are, however, very good, apart from the invariably fatal neo-natal cases. I came to the conclusion that tetanus was not quite the terrifying disease in Fiji that it still proves to be in England.

A round of the wards produced some unusual cases—the woman with burnt soles of her feet from fire walking, and the man extensively burnt from receiving the steam treatment. He appeared to have been overdone and then hidden away, to become septic, lest the Authorities should prove too severe. The Witch Doctor's curse is still a power in these islands and the ability to survive seems to disappear from healthy men and women when this curse is laid on a Fijian.

I never saw a congenital dislocation of a hip, but club feet are common. Anterior poliomyelitis has touched the Fijian islands lightly but has left more cripples in its wake in the Solomon Islands.

Leprosy was, until recently, strictly segregated in leper colonies, and the lovely island of Makogai in the Fiji group was the largest. The island is divided into the clean and dirty areas: in the former live most of the healthy personnel, skilled and unskilled, who run the colony; the latter is the area to which the lepers are confined. The Sisters and the Padrés live in the "dirty" area, and maintain the wonderful atmosphere of courage and hope that pervades the community. The women lepers of all races live in the women's compound, while the men live in villages—the Indian, the Fijian and the Gilbertese villages; the Samoans and the Ellice Islanders also come to Makogai when afflicted with leprosy. There was only one white woman and she had been in the island for fifteen years; Father Choblet, about whom we read in *A Pattern of Islands*, left two or three years ago after twenty-five years' residence. The men tend their gardens, do woodwork and engage in the rather limited opportunity for paid work for lepers on the island. The women engage in excellent needlework, showing the differing designs and work from the different islands in their decorated counterpanes and pillow-cases—one of the latter with the poignant remark

"Hungry for Love". The Leper Trust Board provides all those extras for which the Army rely on the Red Cross; the occupational therapy workshops for men, the needlework room with the battery of sewing machines for women, the cinema for all.

Treatment has been revolutionized by the sulphone drugs, which have healed all but the sores from chronic secondary osteomyelitis or trophic ulcers Plate VI (*a*). The patients are happy and fit, but are not cured with the ease with which the many lesions are induced to disappear. The criteria of cure until recently were two years with no positive smear—now it has been reduced to one year. The swab from the nose, a drop of blood from the ear and a scraping from any area suspicious of a lesion must be free from lepra bacilli over these long periods. These tests are not over-stringent for a cure, as a case may be consistently negative and yet, on operating on a nodular nerve, it will be found to be teeming with bacilli.

The newer approach to leprosy, better called Hansen's disease to dispel the ora of uncleanliness which surrounds the former title, is to allow the patient to return to his village under sulphone treatment and instructed in elementary precautions, once the lesions are cleared up. In the British Solomon Islands, where leprosy is much more common, this is the practice—it appears to be safe, far more humane and allows the medical department to treat a very much larger number of patients from a scattered community, where the full incidence of the disease is still unknown.

The leper colonies in Makogai and in the British Solomon Islands are run by the Sisters of St. Mary, a Roman Catholic Sisterhood drawn from many countries and doing a magnificent work with such enthusiasm and humanity. In Port Moresby, the London Missionary Society runs the leper colony with similar devotion but they are not so fortunate with the siting of their unit. The work of the Churches in these islands shows the co-operation in the past which has minimized the confusion that results from several denominations evangelizing in one community. In the territories mandated under the United Nations, such restraint tends to be interpreted as infringement of liberty and the result is not helpful to unenlightened peoples receiving the Christian message in so many discordant tones. The Tongans are wholly Methodist, the Fijians are very largely Methodist, while the Anglican Communion concentrates on the British Solomon Islands, the Pro-Cathedral of the Bishop of Melanesia being at Honiara. The far-flung Diocese of the Bishop in Polynesia has its headquarters in Suva, although the number of Anglicans in the Fiji Islands is comparatively few. The Roman Catholic influence is strong throughout the territories, partly by reason of the permanence of the missionaries. Those of the older generation were sent out to the South-Western Pacific for life, and the continuing influence of these men and women has been very great. The world has shrunk since those days and the Roman Catholic missionaries no longer go out to the South-Western Pacific for quite such indefinite periods.

The remainder of the article must be devoted to the people and the scenery, with occasionally a little history to complete the picture.

The people of the Pacific are divisible into three groups. The Polynesians ("inhabitants of many islands") occupy the large triangle between Hawaii, Easter Island and New Zealand; I visited Honolulu and Tonga, whose people are Polynesian and are the most highly developed of the races in the Pacific. The Melanesians ("inhabitants of the dark massive islands") occupy those volcanic islands which stretch from New Guinea, through the Solomons, New Hebrides, New Caledonia to the Fiji Islands. These people are Negroid in type, some very small and exceedingly primitive, as in the Solomons, while the Fijians are a fine race and highly developed; a glance at the map will show you that the Fijians have penetrated further into the Pacific than have any other Melanesians; some of their development also, no doubt, results from the considerable intermarriage with the Tongans. Finally, there are the Micronesians ("inhabitants of small islands") who occupy the many small islands north of the equator, including the Gilberts, which I visited; these people show a Mongoloid tendency to a varying degree. It is interesting to see how the three primary races of

the world are represented in the Pacific—the Caucasian, the Negroid and the Mongolian.

Prior to the advent of the missionary to the Pacific Islands there was no written language, and all history was represented by the tales handed down within the tribes. The Polynesians and more advanced Melanesians were organized with chiefs, and even kingdoms, while the more backward Melanesians in the hills of New Guinea still exist in small, closely-knit villages, suspicious of all contact with the outside world and even now hardly ever meeting a white man.

The advent of the white man to the Pacific Islands introduced the idea of sustained and organized work, which was foreign to the custom of these people and to which they reacted, and sometimes continue to react, by what may be described as an attitude of passive resistance. This led the Europeans to import labour, of which the saddest consequence is the large Indian population in Fiji which now outnumbers the native race. It must not be thought that these South Sea Islanders are an indolent people; their accomplishments with Stone Age implements reveal industry and skill; their canoes and some of the fine meeting-houses show a craftsmanship of which they can well be proud, and the Polynesians have some remarkable monuments in stone. Even now, regular work for wages proves unattractive to many who still prefer the village life, which, however, has lost much of its virility in these days of law and order.

These islands boast no indigenous mammals. The wild pigs were probably left by Captain Cook. The fauna is restricted and often the result of importation in fairly recent years.

These South Sea Islanders in former times engaged frequently in wars and indulged in cannibalism. The scarcity of animal protein apart from fish may be a factor in the great prevalence of cannibalism, but there was, undoubtedly, a belief that eating one's vanquished opponent led one to gain his virtues.

THE FIJI ISLANDS

The Fiji Islands were ceded to Britain in 1874 by Thackombau, a Fiji chief at the time in the ascendancy, who styled himself King of Fiji. The story of the Deed of Cession is interesting as it was motivated partly by the need to pay a debt of £9,000 presented by the American Consul for damage to American property on the Islands. There was also the ever-present danger of further wars with the eastern islands under a powerful Tongan chief called Maafu. After America, Britain and Germany had in turn refused to take over the Fiji Islands, a second approach to Queen Victoria was successful and the Deed of Cession was signed by Thackombau and his fellow chiefs in 1874. These islands number 320, but there are only two really large ones; they are volcanic, the main island, Viti Levu, rising to 4,000 ft. in a central ridge. The prevailing westerly winds drop all their moisture on the windward side, which receives a rainfall of up to 120 in., while the eastern half of the island has a much more agreeable climate with a rainfall of only 70 in. Surrounding the islands are the coral reefs, in places four to five miles from the shore, and elsewhere within half a mile, and it was through these treacherous reefs that Captain Bligh, in his overladen open boat from the *Bounty*, managed miraculously to pass, pursued by two canoes manned by Fijian warriors, from whom they only managed to escape when a heavy downpour of rain reduced visibility to a few yards.

The major portion of the islands consists of bush forest, untamed and unproductive of any reasonable timber, occupying country unsuited for any other purpose than timber growing. The narrow valleys and coastal plains grow vast stretches of cane sugar, producing the most valuable export—worth from £5 millions to £7 millions annually. This industry is worked almost exclusively by the Indians and the cane is bought and crushed by the powerful organization known as the C.S.R., or Cane Sugar Refining Co. Ltd. of Australia.

Gold is mined to the value of about £1,000,000 a year, and copra, or the dried

coconut, worth £2,500,000 annually, forms a staple crop, as it does everywhere in the South-Western Pacific.

The people living in Fiji today comprise some 9,000 Europeans, 145,000 Fijians and 165,000 Indians, along with a few thousand Chinese, Euronesians and peoples from other South-Western Pacific islands. Among these latter are the 1,000 Banabans on Rambi Island, and the 80 Ellice Islanders recently transferred to Kioa.

These interesting population movements are the result, in the case of the Banabans, natives of Ocean Island, of the destruction of their island by the quarrying of the phosphatic rock. They acquired sufficient royalties from this industry to purchase Rambi Island in the Fiji group. The Banabans were scattered widely among the Pacific Islands by the Japanese, who occupied Ocean Island between 1942 and 1946, but they were later assembled at Tarawa and moved to their new home in the Fiji group. The Ellice Islanders were moved on account of over-population in their own islands, where the food supply is particularly slender.

The main population is seen to consist of the Fijians, native to Fiji, and the Indians first brought in during the 1880's as indentured labour for the cane sugar industry. As in Natal, they completed their indentures, continued to work hard, brought in their families and multiplied exceedingly until today they outnumber the Fijians and by their earlier marriages, greater fertility and relatively lower infant mortality continue to increase their numerical preponderance. However, their preponderance is not only numerical, their culture is much greater and much older, so they provide many of the professional men in Fiji—the lawyers and the doctors—and possess a good deal of the wealth, although it is very unevenly distributed. They own very little of the land, which soon after the Deed of Cession could no longer be alienated from the Fijians, who, at a disadvantage in so many ways, must yet retain their land vested in the village, tikini or province which form the communal organization of Fijian life. The Indians are well known for their industry, while the Fijian has no long association with work, apart from the communal duties imposed on the villagers by the headman. The Indian has the aggressive striving of the individualist, which leads, perhaps in uncontrolled moments, to some of the more serious trauma in Fiji. It is said that an Indian's worst enemy is another Indian. The Indians live in scattered houses or small family units when engaged in the cane sugar industry, or in the towns where they run much of the business life.

The Fijian is a most attractive character, a cheerful individual of fine physique, given to music and song, friendly in disposition but not given to flights of ambition. When brought up in the Fijian way, he lives in a village, and works in the communal food gardens or on rebuilding the houses as directed by the headman in conjunction with the village council. Crops are grown for consumption and the idea of a cash crop has only recently received attention. Nowadays, a Fijian can pay a tax to exempt himself from communal duties and may acquire land to grow a cash crop, and gain some wealth by his efforts. The Fijian is, however, beset by the dangers of the generous ceremonial of this way of life and much, or all, can be borrowed by relatives, whom it would be quite unseemly to refuse.

Their lives are much enriched by ceremony; the drinking of Yanggona (which the Polynesians call "Kava"), a non-alcoholic beverage obtained from the ground-up root of a pepper tree, plays a big part in Fijian life both on ceremonial occasions of great importance and in the welcoming of any visitor to the village. Distinguished visitors are presented with the Tambua, or whale's tooth, in a ceremony of some duration and detail. The guest may be treated to the spectacle of a Meki or dance, often by men only, and performed seated, the rhythmic swaying of the bodies, oiled for the occasion, and rapid movement of the hands and head, providing a pleasing if unfamiliar spectacle to the European visitor.

The Fijian children are exceedingly friendly and wave vigorously to any passerby, while it is said that when one waves to an Indian child he will look around to see to whom you have been waving.

In appearance the Fijian has thick lips and a rather flattened nose, and yet quite fine and pleasing features. They are very dark skinned with frizzy hair, worn in a mass stretching straight out from the head for 6 in. or more. The hair style was similar for men and women, and until recently this original hair style was essential for a Fijian joining the Police Force. The influence of the Army in the Second World War and more recently in the Malayan Campaign, where the Fijian Battalion played such an outstanding part, has affected the hair style. As many will remember, the Army is somewhat intolerant on this matter and the Fijian hair style had many disadvantages. Many young men have now come to fancy themselves with hair cut short and plastered to the head with a neat parting, and the mop of Fijian hair is becoming less universal, but I was still able to see the Police Force field a Rugby XV, almost every man wearing the traditional head of hair. The Fijian prowess in the Rugby field is well known, the Battalion having an almost unbeaten record in Malaya, where they played the battalions from Great Britain; there is an old-standing rivalry with New Zealand on the Rugby field, and Samoa and other islands take a very active part in a sport that one would not have associated with the South-Western Pacific Islands.

Male dress usually consists of the Sulu—a length of material wrapped round the body like a skirt or kilt. Originally, no doubt, made of the Tapa or bark cloth, it is now of "calico", a term applied to all imported material. Under various names, this unit of dress is universal throughout the South-Western Pacific, although it is naturally gradually being replaced by shorts or something much more elaborate. I was told by a Government Officer in charge of Public Works in the Gilbert Islands that he doubled the work done by his men by putting them in shorts and thus releasing the second hand from attending to the Sulu or "lap lap" as they are called in those islands.

The majority of these pleasant Fijians do not struggle for high attainment but there are a few who have travelled far and attained much distinction in New Zealand or Great Britain, whether in the university or other sphere. These men and women give as much grace and dignity to a gathering as do any of the Europeans, but these Fijians seem as far from the average Fijian villager as we are ourselves, and one wonders how much more able they are to bridge the gap. The women have, of course, been later in travelling and receiving education, so that it has sometimes been difficult for the cultured Fijian to find a wife to share fully in his life. This picture is changing; the Fijian woman is getting a good education and the first woman Assistant Medical Practitioners were qualified last year.

The Fijian, along with all other South-Western Pacific Islanders, had no written language, and the missionaries in first compiling a vocabulary used certain letters which could well be spared to indicate sounds for which we use two letters, an example being "c" to pronounce the sound "th". The sounds "b" and "d" never occur alone, so that "b" and "d" are pronounced "mb" and "nd". The result has been a special value for certain letters applicable only to the Fijian language. This has naturally not been universally accepted and has led to the confusion of two spellings for many place names, e.g. Nandi and Nadi for the airport on Fiji.

TONGA

Tonga was, unfortunately, only visited for a few hours, but I was able to shake hands with Crown Prince Tungi, the Prime Minister; however, I did not meet the Queen. The Tongans have a much fairer complexion than the Fijians and are typical Polynesians with altogether finer features on European standards. They have a fine physique and, with their famous feasts, can become very large.

Nukualofa, the capital of Tonga, is situated in the southernmost group of islands. I was disappointed to find a small modern city laid out in square blocks, like any new-world town. Many of the older buildings in all these islands were of log frames and coconut leaf thatch, so that the past has largely disappeared in the towns and the New Zealand contractors have built a modern town on the flat island of Tongatabu. But more durable monuments have survived in Tonga and witness to the greatness

PLATE VI

FIG. (*a*).

A Solomon Island woman in the Leper Colony. The ulceration has involved the bones, and such secondarily infected lesions do not heal under Sulphone therapy. Her hands were also involved. The anaesthesia relieves her of pain but contributes to the persistence of the deep ulceration.

FIG. (*b*).

Tarawa, The Gilbert Islands.

The village "Water Closets" with tidal flushing twice a day. The approaches are rather public, but necessarily long. Three Gilbertese boys wearing "lap-laps" pose for a snapshot.

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of their ancient kings. The royal burial grounds are quadrangular mounds faced with huge blocks of coral rock, but the greatest monument is an arch of coral blocks, the exposed parts of the upright members being 16 ft. high and the cross piece is 19 ft. long, mortised into the tops of the uprights. The visible parts of this single arch must weigh 30–40 tons. Men skilled in the use of rollers and levers, and with a large body of men at their disposal, must have constructed this arch, much as was done at Stonehenge in our own distant past.

The short time I had in Tonga enabled me only to visit the hospital and meet a few people: I certainly left with the feeling that the visit had been altogether too short, but time was limited and when travelling in the South-Western Pacific the return journey must be carefully worked out when planning any visit, and I found that I had to return on the same plane.

GILBERT ISLANDS

The visit to Tarawa in the Gilbert Islands was for three days and the scenery of these islands, drenched with sunshine, was the South Sea Islands of which I had dreamed. A few degrees north of the equator, it was hot, but the sea breezes always produce a very pleasant climate.

The arrival of the British in 1890, when the islands passed under British rule, interrupted one of the frequent land wars. As can be imagined, the limited land led to much dispute and frequent wars, and it is a regrettable fact that the disputes over land ownership have continued ever since, and since the Second World War yet another Commission has been set up to attempt a solution.

The inhabitants of the islands are Micronesians, small in stature with a slight but definite Mongoloid appearance and dark brown in colour. The Gilbert and Ellice Islands are grouped together and administered together with Ocean Island, but the inhabitants of the Ellice Islands are Polynesians.

There is no running water on a coral island; water is obtained at a few feet under the surface, at places unpleasantly brackish, elsewhere quite pure. It is, of course, filtered from the ocean and lagoon, neither of which can be much more than 200 yards away. The scanty soil on the coral sand or rock supports innumerable coconut trees and a little "taro", a type of root used throughout the Pacific as a staple cereal. Fish form the main source of protein, although nowadays a few pigs and chickens are kept. The only export is copra. The reply I received to the anxiety I expressed on this "single crop" economy was that the Gilbertese can get almost everything from the coconut apart from administrators and doctors, both of which perhaps they might well do without! A sale for the coconut is, therefore, not essential to these islands. The coconut provides the timber and the thatch for houses, the husks and shells for firewood, the coconut meat as food for animal or human consumption, and the contents of the immature nut is excellent for the infant; the spathe can be cut to provide a drink, which, when fermented, gives a very potent draught. What more does one need but the fish on these pleasant islands?

The Native Magistrate was holding Court in the small islet where I stayed and it is the custom for all to dismount when passing the courthouse. The bicycle is a very satisfactory means of transport in an island where the highest point is some sixteen feet above sea level.

The smaller gaols for lesser offences run very economically by a system allowing the prisoners home each week-end; they return with enough supplies to keep them through the coming week.

The scenery is delightful, the lagoons having so many shades of blue and green, the coconut groves and the pure white coral sand completing the setting. Apart from sunsets, there are few reds in the picture. Plate VI (*b*).

I stayed with the Medical Officer, until lately a well-established partner in a practice in Cornwall, but he was only in his early forties and the distant lands seem to have beckoned to his adventurous spirit and here he was, transported to be Senior Medical

Officer in Tarawa, with the late Sir Arthur Grimble's cook (male) in charge of his menage. His wife is soon to join him under the Southern Cross.

The Senior Medical Officer has usually one other Medical Officer with whom to cover the needs of the 38,000 inhabitants of the Gilbert and Ellice Islands, occupying over thirty island units. The difficulties of such a command are better understood when one realizes that each island is an atoll, comprising a string of little islands, never more than 400 yards wide, surrounding a central lagoon which may be ten to fifteen miles across. Transport is difficult to arrange and the only regular service is the recruitment ship from the phosphate industry on Ocean Island, which annually recruits fresh labour while returning those who have worked for the previous twelve months. Medical supervision to such small island units extends the services of the Assistant Medical Practitioner and the Dresser to their full, one Medical Officer being only able to make infrequent visits, while the second has to control the hospital at Tarawa. These medical officers working far from any regular sea or air route were encouraged by the building of a new hospital, now nearing completion: the purchase of equipment and a new X-ray unit was one of the matters engaging their attention. The hospital provides for the minor and major ailments, along with the tuberculous chests or bone disease—as always a major problem in these territories. There was the mental hospital for the uncontrollable case alongside, and the leper community a little way off. There was, fortunately, an English matron and one sister in this hospital, thus assuring the Medical Officer of a standard of nursing which can never be relied upon as yet in the South Pacific without careful European supervision. A "European" dispenser gave valiant support, to complete the hospital team.

BRITISH SOLOMON ISLANDS

From the Gilbert Islands I returned to Fiji, and so via Sydney and New Guinea to Honiara in the Solomons. This trip, hopping from island to island, showed me the innumerable races in this part of the world—short and tall, brown and coal-black. They were, undoubtedly, the most primitive people that I had seen, and there were stories of inhabitants of villages high in the mountains who had hardly ever seen the white man. The population of the British Solomon Islands numbers about 80,000, of which about 38,000 live in one large island, Malaita. Some of the islands by reason of their altitude have less land for native villages, but that does not appear to be the primary reason for the congestion in this island of Malaita. Since the war there has been considerable resistance to the European rule in this island, as also in New Guinea, but more co-operation has been forthcoming in the last few years. The economy relies entirely on the coconut, although geologists are active in the search for mineral wealth in these volcanic islands. The names in this part of the world recall the Japanese war—Guadalcanal, Rabaul, Lae, the Owen Stanley Mountains reaching 23,000 ft., and Port Moresby—and there are very frequent reminders of this recent war in the wrecks that are scattered around the coasts, the rusting machines of war, the many aerodromes and some large cemeteries. For many years after the war, scrap iron was the most valuable export from the British Solomon Islands. Rehabilitation of the coconut plantations is now far advanced and the coconut again dominates the scene.

Honiara is the capital of the British Solomon Islands and has a rapidly growing community. Here live most of the Government Officers and their families, but the European community remains small. There are, for instance, only four doctors in the service, two living in Honiara, one largely controlling the hospital while the senior is the Senior Medical Officer with the administrative and public health work as his main preoccupation. There is a fair sized Chinese community in Honiara, running many of the shops and other small businesses. Honiara was selected as the capital after the War, the American buildings, aerodromes and roads providing sufficient inducement. The absence of any deep-water port in the vicinity, with the consequent reliance on lighterage for unloading every ocean-going vessel is, however,

proving a serious drawback to the siting of the administrative centre and only town on the islands.

After a week in Guadalcanal, I retraced my steps as far as Port Moresby, the chief town and administrative centre of Papua—New Guinea. Here, 6,000 Australians and New Zealanders enjoy complete absence of Income Taxation, an inducement sufficient to encourage many to accept the less pleasant climate and more pioneering conditions in these Territories, administered by Australia under a United Nations Trust. At the invitation of the Medical Department, I stayed there three days and saw the tuberculous spines and the leprosy cases which presented orthopaedic problems.

The newspapers at that time carried news of raiding by some primitive mountain races with strong suspicion of cannibalism. It appeared that it would take some two weeks to penetrate into the area in order to attempt to obtain the facts of the situation. The country is mountainous and hostile, the people are very primitive and often warlike, and civilizing influences are only just touching them. Australia struggles with the many problems involved and is somewhat unsympathetic to suggestions from the United Nations that a date should be given when Papua–New Guinea will become a sovereign state.

Nine weeks in the South-Western Pacific were drawing to a close and I left for home, very grateful for the opportunity of seeing so much and hopeful that one day I might be able to return to these beautiful islands and colourful peoples.

THE PREVENTION OF ACUTE RHEUMATISM*

BY

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If the title of this paper suggests that the mystery of acute rheumatism is solved, it would be far from true, for the factors which condition the occurrence of this disease still elude us.

SOCIAL INCIDENCE

It has long been known, however, that acute rheumatism is a disease which shows a striking social incidence. The researches instituted in Bristol by Dr. Carey Coombs confirmed, in 1930, that it was predominantly an urban as opposed to a rural disease, the incidence in Bristol being about four times that in the surrounding counties[1]. In Bristol itself there was an even greater variation in different parts of the city which we were later able to show was clearly related to both overcrowding and poverty[2, 3, 4]. Which of these is the more important we do not know. Overcrowding might be the crucial factor by facilitating infection, or poverty might result in a deficiency of some essential food factor. It may be that the recent change in social conditions is responsible for the rapid decline in incidence and severity of acute rheumatism which has taken place during the last thirty years. In 1925, according to the Registrar General's returns, rheumatic fever was responsible for 1885 deaths in England and Wales. whereas in 1955 the comparable figure was 227. The same trend is revealed by figures obtained from notifications of acute rheumatism. Since October 1947 the disease has been notifiable in Bristol and certain other parts of the country. In 1948, the first full year of notification, there were 89 confirmed cases of acute rheumatism notified in Bristol for every 100,000 school-children; in 1956 the incidence had fallen to 27.5 per 100,000.

STREPTOCOCCAL INFECTION

We should, however, perhaps note that this decline is paralleled by a decline in the severity of streptococcal illnesses, for instance deaths due to scarlet fever. For it is now as certain as anything can be in medicine, that the Lancefield Group A beta-haemolytic streptococcus is the precipitating cause of rheumatic fever. How the streptococcus causes rheumatic fever is still obscure and why only ½–5 per cent. of persons afflicted with a haemolytic streptococcal infection subsequently develop rheumatic fever, and why children aged 5 to 15 do so more frequently than adults or younger children remains one of the major mysteries of medicine. But we can now say "No haemolytic streptococcal infection, no acute rheumatism".

The evidence for this is four-fold. First, from clinical observation there is the now well-known phenomenon of a streptococcal sore throat followed after a "silent" interval by rheumatic fever; this has been particularly clearly studied in the long-term hospital schools for rheumatic children. Secondly, streptococcal antibodies have been recognized in the blood of patients with rheumatic fever. Todd first described how a high titre of anti-streptolysin O occurs as frequently in patients with acute rheumatism as it does in patients convalescent from scarlet fever; there is a similar rise in the titre of the other antibodies—anti-streptokinase and anti-hyaluronidase. Thirdly, there is the evidence obtained from epidemics of rheumatic fever; these have been observed in schools, and training depots where young people are crowded together, and they are

* From a paper delivered to the Bristol Medico-Chirurgical Society on 9th January, 1957.

always preceded by an epidemic of haemolytic streptococcal infections. Finally, we have learnt that if we prevent haemolytic streptococcal infections we abolish recurrences of acute rheumatism.

PREVENTION OF RECURRENCES

It is well recognized that acute rheumatism is a disease in which recurrences are very common and that the rheumatic patient's greatest risk of such a recurrence is during the first few years following the initial attack. This gives us our first clue as to how we should tackle the problem of prevention. If we could prevent streptococcal infections in rheumatic children for five years after the initial attack or during school life we should prevent the majority of recurrences, and it is of course the recurrences that are of major importance in producing serious and crippling rheumatic heart disease. There is now ample proof that this is possible in either one of three ways.

1. *Prophylactic sulphonamide*

We can keep all children who have had acute rheumatism on continuous prophylactic sulphonamide, and this has been done on the largest scale for the longest time. Published results up to date have recently been summarized[5]. In nearly fifteen hundred "patient years" the incidence of rheumatic recurrences was reduced to nearly a tenth of that in carefully matched untreated controls. But sulphonamide prophylaxis is not without its hazards. It may, fortunately rarely, produce skin rashes and blood dyscrasias; these usually occur in the first few weeks and most people therefore recommend that prophylactic administration of sulphonamides should be started while the patient is in hospital with the acute attack of rheumatic fever so that he can be under close observation during the most critical time. In a small experiment we did some years ago we found that we were afraid to continue to give the drug in a certain proportion of cases because of skin rashes and changes in the blood count—although we have since been told that we were too timid. Also we found that treated patients did not gain as much weight or seem quite so well as controls. Finally there is some—perhaps slight—evidence for the risk of resistant streptococci emerging. But the method is simple and cheap, suitable dosage being 1·0 g. of sulphadiazine daily or 0·5 g. for a child under seven. It needs, of course, full co-operation of the patient so that he never misses a dose, because one day without the drug is quite sufficient for streptococcal infection to occur with a subsequent relapse of acute rheumatism.

2. *Oral penicillin*

For this reason attempts have been made to use other measures and here penicillin has proved to be unequalled. Penicillin may be used by mouth in the shape of tablets of Penicillin G, 200,000 units twice daily, or perhaps more efficiently of phenoxymethyl penicillin (Penicillin V) 120 mg. twice daily. Of course penicillin prophylaxis of rheumatic recurrences has a much shorter history and has been used less extensively than sulphonamides, but the results are impressive[6, 7, 8]. The collected figures for seven reported series shows an incidence of 8·7 per cent. recurrences per patient year in controls and only 0·6 per cent. in treated children. There is the same problem about co-operation of the patient, but toxic effects are extremely rare and penicillin-resistant haemolytic streptococci have never appeared. There is a risk of producing penicillin-resistant straphylococci but these organisms are now so widespread that the danger is perhaps not serious.

3. *Penicillin injections*

A different approach is to administer long-acting repository penicillin—benzathene penicillin—by monthly intramuscular injection. An injection of benzathene penicillin (Penidural L–A) 1·5 mega units (5 ml.) once a month produces blood levels which are adequate for prophylaxis. It has the disadvantage of all injections in that they may

produce local pain and general malaise and fever, but with the new preparation introduced last autumn the incidence of these unpleasant side effects has greatly diminished. Moreover, results are perhaps better than with oral penicillin[9]. Incidentally, while sulphonamides are of little value in eradicating the haemolytic streptococcus from carriers, penicillin by mouth or injection is extremely efficient, provided it is continued for long enough; a monthly injection of benzathene penicillin is perhaps the most effective of all[10].

Duration of Prophylaxis

We can thus prevent rheumatic patients from developing recurrences.—but for how long should prophylaxis be continued? Some enthusiasts in the American Heart Association say for life. This may prove in the end to be necessary but it is a severe sentence. Perhaps a wiser plan at the moment is to advise some form of continuous prophylaxis for five years after the last attack of acute rheumatism or until the age of 15 years, whichever is the longer. If so we should always remember to re-institute prophylaxis whenever the patient is exposed to excessive risk of infection with the haemolytic streptococcus. These occasions are:

(1) When any individual in the patient's household develops a haemolytic streptococcal infection.

(2) When the patient is admitted to any hospital ward.

(3) When he enters any semi-closed community such as a camp or institution where haemolytic streptococcal infection is likely to be prevalant.

Such intermittent prophylaxis for special exposure might well be maintained for life.

PREVENTION OF FIRST ATTACK

So much then for the prevention of recurrences. Can we do anything to prevent a patient who has a haemolytic streptococcal sore throat from subsequently developing acute rheumatism? Our evidence that we can is largely derived from work carried out on the U.S. armed forces, but it is fairly conclusive. Early results were encouraging and indicated that any treatment which rapidly and completely eliminates the haemolytic streptococcus from the throat of the patient, provided that it is instituted sufficiently early in the course of the disease, will prevent subsequent acute rheumatism[11, 12]. How can we do this? It requires more than merely "treating" the sore throat or even suppressing the organisms. Sulphonamides for this are useless both in preventing rheumatism and in eliminating the streptococcus. Penicillin properly given is very effective; inadequate dosage is, however, as useless as sulphonamides. A single dose, however large, of a short-acting preparation suppresses the streptococcus for a time only. What is needed is adequate blood levels for ten days. How we are to achieve this depends of course on the type of penicillin we use. Oral penicillin is effective for a few hours, procaine penicillin for a day or so, procaine penicillin in oil for some days, and benzathene penicillin for weeks.

Penicillin dosage

We can thus gain our end equally well (*a*) with large doses of oral penicillin (Penicillin G, 200,000 units four times a day, or Phenoxymethyl penicillin—Pencillin V—120 mg. four times a day) for ten days, but not if we use an inadequate dose or give it for too short a time; (*b*), by several injections of procaine penicillin in oil (procaine penicillin in aluminium monostearate 500,000 units every other day for four doses); or (*c*), by one large injection of benzathene penicillin, 1·5 mega units (Penidural L.A. 5 ml.)—or perhaps preferably a combination of benzathene with crystalline and procaine to give high initial levels, for instance 300,000 units of Penicillin G, 300,000 units of procaine penicillin and 600,000 units of benzathene penicillin (Penidural all-purpose). And we can use the same measures to prevent a recurrence after a sore

throat in a rheumatic child who has not received prophylaxis or where prophylaxis has for some reason or other failed. In rheumatic children where there is a 50 per cent. risk of a streptococcal sore throat giving rise to a relapse of acute rheumatism the results of this prophylaxis are very encouraging[13]. In one of the earlier studies half the controls had a relapse and only 6 per cent. (two) of the treated cases, and both of these had a relapse of the streptococcal infection, i.e. the organism was probably not completely eradicated. In a series of patients with sore throats some of whom received no treatment, some sulphonamide and some penicillin, even when treatment was delayed until the ninth day of the illness, only 3 of 420 patients treated with penicillin developed rheumatic fever, i.e. 0·7 per cent., whereas 3·6 per cent. of 220 controls and 5.2 per cent. of 230 treated with sulphonamides developed rheumatic fever subsequently. Is there any alternative to penicillin if perchance a patient is sensitive to penicillin? Nothing so effective although the tetracyclines have some use as shown by Catanzaro and his colleagues[14].

Recognition of streptococcal sore throat

If we are then, to treat efficiently all cases of haemolytic streptococcal pharyngitis in order to prevent first attacks of acute rheumatism, how are we to recognize them? Clearly we do not want to employ such energetic therapy for all and every sore throat of which perhaps 80 per cent. will not be due to the haemolytic streptococcus. A throat swab is of course a help, but the presence of haemolytic streptococci in the swab does not prove that they are the causal organisms as up to 25 per cent. of normal children are carriers. On the other hand, a negative throat swab, provided it is taken properly and cultured without undue delay, almost certainly excludes haemolytic streptococcal infection. For these reasons the clinical features become important, and some people think that it is possible to diagnose a streptococcal sore throat accurately in a fair proportion of cases on clinical grounds. The important features are:

(1) The rash of scarlet fever.
(2) A red oedematous throat with exudate and enlarged cervical glands.
(3) The occurrence of abdominal pain, headache and vomiting.
(4) If a white blood count is possible, a polymorphonuclear leucocytosis.
(5) The existence of haemolytic streptococcal infection or its complications—e.g. otitis media—in the patient's household.

Features which make a haemolytic streptococcus unlikely as the cause are the occurrence of coryza, cough or hoarseness. If in doubt perhaps the wisest course is to take a throat swab and have it plated as soon as possible and to start treatment with penicillin but to continue only if the swab proves positive.

SUMMARY

To sum up then, to prevent relapses of rheumatic fever we can employ continuous prophylaxis with sulphonamides, or with oral or injected penicillin. This must be continuous and should be continued for five years after the last attack or until the patient is 15, whichever is the longer. This treatment should be re-instated later in life whenever the patient is exposed to special risk. We can hope to prevent first attacks of rheumatic fever and relapses where prophylaxis has failed by adequate treatment of all haemolytic streptococcal infections so that the organism is eradicated from the throat. This we can achieve by oral or injectable penicillin in doses that will give an adequate blood level for at least ten days, or if this is contra-indicated with a tetracycline, also for ten days.

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HEAD INJURIES*

BY

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The management of cases of head injury has been improving since the turn of the century; but in this mechanical age their incidence has risen steeply.

One major advance in our understanding of the mechanism in head injury we owe to the writings of Sir Geoffrey Jefferson before the War. He brought to general notice the importance of midbrain dysfunction in the causation of unconsciousness, not only following trauma but also in cases of tumour and abscess of the brain. This is how it comes about. When one cerebral hemisphere is displaced medially—as by clot on the convexity, or by pus or fluid, for that matter—or when it expands by reason of a swelling within it, only a limited amount of room to contain its increasing volume can be afforded by bulging across the midline under the falx. The only other available direction for expansion is downwards through the tentorial aperture. The innermost part of the temporal lobe, adjacent to the midbrain, becomes squeezed round the free margin of the tentorium, forming a hernial ridge which compresses the midbrain and also stretches the third cranial nerve. The brain, then, has to expand downwards through the tentorial aperture. When it does so it also pushes the midbrain and pons caudally. The effect of this movement is to stretch the many small arteries passing in to nourish the midbrain and pons. The basilar artery is tethered by the posterior cerebral arteries, and its caudal movement is therefore limited. The midbrain and pons suffer an ischaemia and consciousness is lost.

MIDBRAIN HAEMORRHAGE

I have dwelt a little on the morbid anatomy of these displacements of the brain, which incidentally are common to all types of expanding supratentorial lesion, because the next step in the story is an irretrievable disaster. Plate VII shows the disaster—acute softening and haemorrhages within those vital parts of the brain. We know that it can occur quite suddenly, revealed in those cases of frontal extradural or intracerebral clot, and occasionally of abscess too, where the patient remains conscious, until he plunges into coma. Nothing can save the patient with haemorrhages in the brain stem. But in most cases there is ample time for investigation and intervention, provided the earliest clinical signs of impending danger are heeded. More of that later.

It is a common misconception that a high pressure within the head causes drowsiness or unconsciousness. If saline be injected into the lumbar theca experimentally to produce a pressure of 1400 mm. or more, the chief symptom, if any, is dizziness. That is a pressure far exceeding those found even in the desperate cases.

Other factors in the improving state of affairs in the handling of cases of head injury have been, first the additions to the operating facilities in the country as a whole, whereby it is easier for burr holes to be made in the skull when there is doubt. Secondly, the introduction of arteriography has been a great advance. Arteriograms show the displacements of arteries occasioned by the presence of clots or of post-traumatic cerebral oedema. They give precision to diagnosis, or to what has perhaps been mere clinical conjecture. This applies particularly to those patients who are unconscious and quite "inaccessible" from the moment of injury. They have cerebral lacerations or severe contusion and may be suffering from the mounting effects of intracranial bleeding or of swelling of the brain. In either event the outcome is likely to be fatal

* A paper given to the Bristol Medico-Chirurgical Society, 13th March, 1957.

unless operation is performed for evacuation of clot or to provide a generous decompression. Those cases used to languish and perish, often with the label of "cerebral irritation".

With the ophthalmoscope one can see at once if the intracranial pressure is raised, because the retinal veins can be seen to be engorged. It takes a day or two for papilloedema to develop, and very often that time is not granted. This venous engorgement of the retina is probably an immediate indicator of mounting intracranial pressure.

In hospital the intracranial pressure can be measured by lumbar puncture and manometry, but this can be a dangerous procedure if a large expanding lesion be present and the cerebellar tonsils are consequently jammed down into the foramen magnum. Indeed in those circumstances a falsely low manometer reading may be obtained. The ophthalmoscope is helpful in sorting out those cases in which lumbar puncture is certain to be safe.

But lumbar puncture may have to be used as a treatment for high intracranial pressure in some cases after a head injury, when the cerebrospinal fluid contains blood. In such cases it is presumed that red cells get wafted towards the Pacchionian villi in the pulsating slow current of subarachnoid fluid, and that they pack around the villi, interfering temporarily with the resorption of the cerebrospinal fluid into the venous blood-stream. Lumbar puncture relieves the severe headaches of which these patients complain, but it is wise to know beforehand from an arteriogram that no clot or swelling of the brain is present.

THE NATURE OF CONCUSSION

Some remarks are appropriate now on the subject of simple concussion, that brief reversible coma, unassociated with structural damage to the brain. Concussion remains a mystery. It would be true to say that we have no more concise ideas as to its nature than our medical ancestors who in their Latin texts called it *commotio cerebri*.

Professor Dott of Edinburgh has recently advanced a suggestion which may well be right on the target; I am greatly indebted to him for allowing me to mention his hypothesis.

First of all I should say that cerebral arteries resent stretching, and respond by contracting vigorously. Some clinicians still find it hard to believe that the calibre of the intracranial arteries is variable; the neurosurgeon sees this occasionally to a quite dramatic degree.

The second point supporting the argument concerning the theory of concussion is that the unconsciousness of subarachnoid haemorrhage due to rupture of an aneurysm at or near the circle of Willis is almost certainly due to spasm of the perforating arteries which run up from the circle *via* the perforated spots into the base of the brain. We deduce this from studies of the territories of red softening in the diencephalon, apparent in fatal cases of subarachnoid haemorrhage, devoid of thrombosis in the arteries supplying the softened territory; this is an important observation. The diencephalon is another "seat of consciousness", to put it rather colloquially.

A further point is that the timing of the unconsciousness in very many cases of spontaneous subarachnoid haemorrhage is just right for the onset of a vascular spasm. How often do we hear that the patient had time to cry out "Oh my head" before he or she drops to the ground. Moreover, the volume of blood which escapes from the aneurysm is in so many cases patently far too small to cause unconsciousness and death.

Professor Dott postulates that a shearing strain is imposed on those small perforating arteries at the base of the brain by a concussional blow on the head, and that the vessels promptly go into spasm. The quite surprising mobility of the brain within the cranium certainly permits such a hypothesis. His postulate of a temporary diencephalic ischaemia could account for the clinical features of simple concussion and for its variably rapid reversibility.

RADIOLOGICAL EXAMINATION

In these days it is perhaps unnecessary to stress the importance of radiological examination of the skull. The benign-looking scalp wound in a patient who has perhaps not even been stunned, may have a depressed fracture underlying. Sometimes little tufts of hair get trapped in a fissure fracture, which must have received the hair when the crack was sprung open at the moment of impact. Cases like this, treated casually, can lead to endless trouble and danger with osteitis and its intracranial sequelae.

Many years ago a patient was admitted to a ward of my previous hospital following a car accident. She had been dazed for only a very short time, and had only a few abrasions. I had to insist that the skull be examined radiologically before the patient be allowed to depart. The film showed an upholsterer's tack driven squarely into one parietal bone, and with not a drop of blood to show for it on the scalp.

Medico-legal attention is far too much focussed on the supposed importance of fracture of the skull. Undoubtedly this stems from the obvious importance of fractures in the limbs and trunk, in regard to weight-bearing and other uses of the limbs. Of course one cannot belittle the fracture which could provide a pathway for intracranial spread of infection.

Here is another story. Some years ago a man sustained a head injury and had an unimportant but clinically-obvious fracture of the base of the skull anteriorly. He was a known bleeder but his blood seemed normal with all tests then current. He developed an intracerebral haematoma. We operated, and as we had warned the relatives might well be expected, the haematoma recurred in spite of the most careful haemostasis. Eventually we lost this patient after re-operation and transfusions, and the relatives were satisfied that we had done everything possible. At the inquest the main cause of death was given as fracture of the base of the skull. The family were aghast, and obtained free legal aid to try to make out a case of negligence. The X-rays, including our arteriograms, have I suspect been around the country to various experts —all at the public expense—for someone perhaps to detect the invisible fracture which was of such negligible clinical importance in the case.

Per contra, one sees cases with a mosaic of fissures criss-crossing over the vault. I can say from long experience that among those surviving for admission to a neurosurgical service, it is rare to have to operate for intracranial mischief in cases of this type, and one after the other they recover—most of them completely. One must presume that much of the force of impact is expended in fragmenting the skull, and that some favourable deceleration of the brain can give it some protection.

So much for the financial value of fractures of the skull. It may be objected that the presence of a fracture is an index of the severity of injury, but it is an inconstant and unreliable index. Some of the worst post-traumatic disabilities follow injuries without fracture. The attention of legal assessors ought to be concentrated on the story of the injured person's unconsciousness and its sequelae, and on the retrograde and post-traumatic amnesia.

CHRONIC SUBDURAL HAEMATOMA

I think it appropriate to say a little about chronic subdural haematoma. I wish first to explode the idea that a history of relevant head injury is usually forthcoming in these cases. In fully half of the cases I have handled, no knock on the head was known to have occurred. Many of these patients, seemingly with a reasonably good recollection of events prior to operation, remember the pertinent injury as they are about to go home from hospital, or tell us about it at a follow-up appointment. Often the causative blow on the head is seemingly trivial, and is therefore forgotten.

The next point worth emphasis, is the frequency with which *fluctuation in the level of consciousness* is observed in this condition. This is an important diagnostic feature; and it has a plausible explanation. The most gross examples of tentorial herniation are to be seen in the presence of a chronic subdural haematoma, because time is afforded

for a gradual deformation of the parts. I have seen part of the temporal horn of the ventricle in one of these herniations. Now, compression of the midbrain must impair the flow of fluid down the aqueduct, and the lateral and third ventricles dilate. It is probable that fluid is forced past the obstruction intermittently, which would accord with the frequently-varying grades of consciousness so characteristic of chronic subdural haematoma.

In earlier years, before the War, we used to have an unexplainably high mortality with these cases. We would let out a large volume of dark brown fluid, the unconscious patient would recover in the theatre—and perhaps even ask for a cigarette (which note you, in those carefree days, was provided *instanter*—match and all!) Next day drowsiness would recur, and by midnight perhaps we would be at him again with brace and burr. Some more fluid would be released, with a shorter and less effective reprieve; but surely he died. It was all a bit odd, and highly frustrating. Mr. Dott had us revive the Boston practice in Dr. Cushing's clinic, of perfusing the carotid arteries with formol-saline some hours before the autopsy. This procedure of partial fixation of the brain *in situ* reveals the distortions and displacements of the brain to be far more gross than is apparent when the brain is removed in the unhardened state.

Returning to our problem, there was the solution plain to see—the unrelieved temporal lobe herniation and acqueduct obstruction, with secondary dilatation of the ventricles.

Our mortality rate dropped abruptly when we drained one lateral ventricle continuously into a glass flask by catheter and Southey's tubing for a few days to give time for disimpaction of the herniation to take place. A much more direct and simple remedy was proposed by Lalonde and Gardner of Cleveland (1948)—merely to push the herniated ridge of temporal lobe from below upwards by injecting saline solution into the lumbar theca. This is dramatically effective.

In this connection, there is an old and true adage concerning cases of extradural haemorrhage, that if the brain does not "expand" when the clot has been evacuated, the patient will die. In reality there is nothing of expansion in the situation; the brain is held in its displaced position because the temporal lobe is nipped in the tentorial aperture and cannot come up to its normal level in contact with the skull. This trick of intrathecal saline injection has diminished the mortality in cases of extradural haematoma also.

THE SIGNS OF DANGER IN HEAD INJURY

I come now to the "meat" of my discourse. It concerns the suggestions I should like to offer for guidance in suspecting that trouble may be on the way in a case of head injury.

I think the commonest, earliest and most significant sign is a *deterioration in the state of consciousness*. The lucid interval syndrome of extradural haemorrhage is a classical example: first the concussional unconsciousness, then recovery—and often complete recovery—then headache and probably vomiting, drowsiness and then unconsciousness, as the midbrain distortion develops.

But what about the patient with a contused brain and perhaps with a laceration, who is unconscious from the start? He too shows a lessening of responsiveness, if he is running into trouble. Incidentally, I recommend firm rubbing of the skin over the sternum as a most effective means of penetrating the screen of unconsciousness.

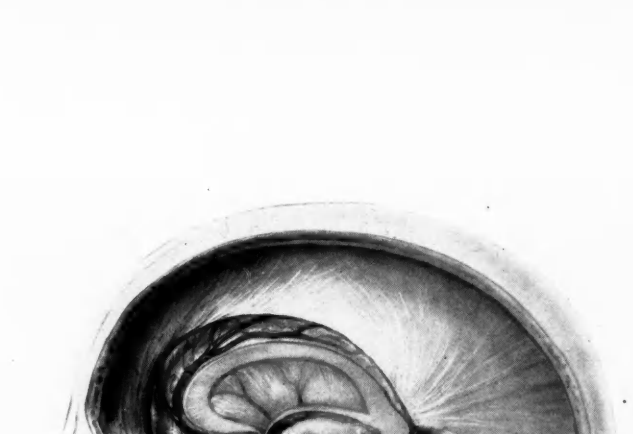
Another important clue is the appearance *de novo* of neurological abnormalities, or the increase of existing deficits, such as paresis, changes in reflexes, or dysphasia if the patient has previously been able to speak. This may signify that an intracerebral clot is enlarging or that oedema is developing around an injured area of the brain.

Dilatation of the pupil on the side of the expanding lesion is an ominous sign, universally familiar. Plate VIII shows why this occurs. Once again the herniation of the temporal lobe is the cause of the trouble. If both pupils dilate and become inactive to

PLATE VII

A section through the midbrain showing widespread punctate and diffuse haemorrhages, which were the consequence of unrelieved supratentorial hypertension and caudal displacement of the brain stem.

PLATE VIII



(From Thompson and Miles "Manual of Surgery" 1939: by courtesy of Oxford University Press, and the Editor, "The Black Bag").

The proximal hemisphere has been removed, exposing the uncompromised third nerve on the same side. Beyond it the opposite third nerve is seen, stretched in a curve round the herniated uncus of the temporal lobe on the side of the expanding lesion. The caudal displacement of the pons is indicated by one of the arrows and the mechanism of traction on the sixth nerves is figured.

light soon after injury, the midbrain may be assumed to have gone out of commission, and the patient's fate is sealed.

Restlessness is a common accompaniment of intracranial hypertension. There is a quality about this restlessness which becomes familiar to us who see many of these patients, but which I find rather hard to describe. It is intermittent though frequent, and has a sort of frantic determination about it. A full bladder is, of course, another and banale cause of restlessness; and retention of urine is not uncommon after injury to the head.

Another portent of danger is a *deepening of respiration.* I do not refer to the familiar deep, rapid and noisy breathing which so often advertises to the inmates of a ward that serious events are afoot behind the screens. You must all have seen the patient who has a pink suffusion of his face and who is breathing as if he had been engaged in some moderate exertion. He looks hot—and in the vernacular, he certainly is a "hot" case.

It would, I think, be redundant for me to do more than enumerate the other classical accompaniments of severe intracranial hypertension; the rise of blood pressure, to keep the cerebral circulation going in a compressed brain; the slowing of the pulse and its ante-mortem acceleration; the slowing of respiration and its ante-mortem acceleration. Cheyne-Stokes respiration is of ill omen; when occurring early, it indicates that a mortal injury was sustained at the time of the accident. If it appears in a case for which it seems that something might still be done, it usually means that matters have gone too far for any effective treatment.

THE SELECTION OF CASES FOR NEUROSURGICAL TREATMENT

I should like now to raise the question of the management of head injuries in a topographical sense.

In the first place only about one in eight cases of acute head injury admitted to hospital requires opening of the head, either by burr holes or by craniotomy.

Circumstances beyond our control have enforced a "vetting" of cases for transfer to the neurosurgical service in Bristol. We have had to ask to be allowed the opportunity of discussion by telephone before accepting cases. We welcome this opportunity of expressing our gratitude to the general practitioners especially and also the consultants, in the Region, for the apparent willingness with which they have put up with this innovation. Of the 650 or more cases admitted annually to the Unit, about 400 are emergencies—or "urgencies"—admitted within one or two days: and of these acute cases only one in five is a case of head injury. We are greatly helped also by the readiness of consultants to accept back their cases for completion of convalescence or if the case is inoperable; but all concerned would be more happy were we able to "see our own jobs through".

Extradural haematoma is not at all rare—we have twelve to sixteen of them a year. Acute subdural haematoma occurs more frequently, and swelling of the traumatised brain requiring decompression, less often. Some years ago a questionnaire was sent round by me to other neurosurgical centres, using the incidence of cases of extradural haemorrhage as a yardstick. The incidence in Bristol was three to ten times higher than in nearly all of the other centres. Now I cannot believe that this Region is a particularly dangerous one in which to live, and I am led to the conclusion that those telephone consultations must be bringing along the cases.

Mr. Phillips and Mr. Hulme have been looking into our archives and several pertinent facts emerge. The first is the conspicuously low mortality-rate among the cases of extradural haemorrhage: in some recent years it has been as low as 10 per cent. In America it is about 45 per cent. in neurosurgical centres, and around 75 per cent. in general hospitals (Kahn *et al.*, 1955). These figures do not reflect on the standards of diagnosis and treatment, but bear directly on what goes on before the patient reaches hospital, and how quickly action is taken. The alert practitioner—and we seem to have a lot of them around—the houseman who is "on his toes" and a generally commendable ambulance service, are all playing the major part in the results.

Sometimes, on discussion, it has been very evident that operation must proceed forthwith at the peripheral hospital.

The second point which Mr. Hulme has brought out in his enquiries relates to the end-results in acute subdural haematoma. Because of the associated contusion and laceration of the brain, the proportion of complete recoveries, in the very few published series, has been notoriously low. The return-to-work rate in our cases has been improving over the years, and is now around 35 per cent.—a very good comparative figure. There can be no doubt that this beneficent trend follows from our receiving these cases earlier, and I think the telephone is helping in this. The results in cases of extradural haemorrhage present a contrast; nearly all recover fully. They are therefore economically the more rewarding.

Some apology is required for the note of domestic advertising in those comments on our experience in Bristol, but the evidence presented indicates directions in which improvements could be effected. There is no magic in what we do for those head-injured. It is traditional treatment and some of it was carried out successfully in general hospital services—and still is—throughout the country.

NEUROSURGERY AND THE GENERAL SURGEON

Having regard to the fact that most of our cases in which a major intracranial lesion has been revealed at operation have come from this area, within a radius of thirty miles or so of Bristol, the question arises, what of the similar cases at a distance from a neurosurgical centre? My own view is that ideally in each major centre of surgery at a distance from a neurosurgical unit it would be desirable for two of the general surgeons to be versed in the tricks of the trade, particularly in the staying of haemorrhage during craniotomy, and in haemostasis of the brain.

It is no longer sufficient to rely on the classical subtemporal craniectomy for middle meningeal haemorrhage. One third of the clots we evacuate are frontal or parietal, and would be missed by the classical operation, which is also often useless and dangerous in the other conditions requiring decompression.

Even more to the point for the surgeon is the acquisition of speed in the preparation of the scalp and in raising a bone flap, for the patient's sake at times, and for the sake of the theatre and other staff at all times. It is largely a matter of providing good razors, proper bone-cutting tools, diathermy of a quality suitable for intracranial haemostasis, and a sucker which will stand up to hard work. But there is more to it than operating. The diagnostic side would need development, and in that arteriography would play a part. Cerebral angiography is spreading. It has ceased to be the monopoly of the neurosurgeon, now that its technique has been radically simplified.

I can see all sorts of difficulties in trying to implement a policy of development of some neurosurgical knowledge and skill at the periphery, even were the policy accepted in principle. The optimum grade in which a training of several months could be given would I think be in that of senior registrar. But then the difficulty arises of ensuring a distribution of those trained surgeons. Perhaps secondment to a neurosurgical unit, for the requisite time, on appointment (in advance) as a general surgeon would overcome the difficulty. I must emphasize that a casual or brief introduction to neurosurgical diagnostic and operative techniques would be ineffective in its purpose and potentially dangerous. Further, the suggested policy should apply only to those cases of such urgency that time will not permit of their transfer to a special unit.

I see no difficulty in disseminating knowledge about better after-care, which is dominantly a matter of preventing inhalation and infection in the airways.

PREVENTION

I should like to draw attention to a book which records the proceedings of the first Congress on the Medical Aspects of Traffic Accidents, held in Montreal in 1955. The contributors are from Canada, the United States and this country. It is published

under the aegis of a Trust Foundation in Canada. Copies have been given to medical libraries (including that of Bristol University). Slow-motion film analyses of what happens to car and occupants in different types of crashes are described. The anatomy of wrecked cars is studied, as are tests of safety devices and of radical alterations in car design and in fittings. The law relating to "drunk-in-charge", and tests for drunkenness, are discussed in the light of the various practices in different states and countries. And as an example of other topics, the difficult question of the controlled epileptic and his driving again.

I noticed recently in Montreal that the driver and passengers fastened the aeroplane type of safety-belt which was fitted to the seats in many of the private cars, even for short journeys in the city.

The high cost to the individual of hospital treatment in the New World, which commences with a charge for the ambulance (complete with intern and orderlies) from the scene of the accident, must be an incentive to lessen the toll of the roads. A costly draft on an annual benefit under personal sickness insurance, may leave a debt with nothing to cover illness. Damages awarded in the courts may not be recovered for years, if at all. In contrast, we are fortunate in this country, with our hospital service which has *always* been free; and with our compulsory third-party insurance which has no ceiling on company's liability, and is not therefore tied to the annual premium the driver elects to pay, as in the New World.

It is of interest that we have observed a substantial drop in the incidence of cases from motor-cycle accidents admitted to Frenchay Hospital since the wearing of crash-helmets became common. As might be expected, our plastic-surgeon colleagues have not noticed any lessening of their load of facial injuries; indeed some of the injured are probably now being spared from death at the roadside, and reach hospital.

A large dividend can be expected from research into the causes of accidents and into ways and means of minimizing the injuries received in them. If due regard is paid to the latter factor we shall have to accept some sacrifice of style and elegance in the motor-car of the future. The brain of civilized man is becoming more widely of importance to his earning capacity and to his place in society. It behoves us to place the conservation of brains in the forefront of our considerations.

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BRISTOL AND BORDEAUX

BY

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For the past ten years relations between Bristol and Bordeaux have been fostered by the Bristol–Bordeaux association, which, among other things, links the schools and the universities in the two cities. The association's main function is the organization of the exchange of numbers of school-children for visits of two weeks or more each summer. Individual visitors are also exchanged between the universities, and recently lecturers have been exchanged between the faculties of medicine. In April 1956 I had the pleasure of visiting Bordeaux for a week, when, in return for two lectures given to medical and student audiences, my wife and I were warmly and hospitably received by members of the medical faculty in their hospitals and departments, and in their homes. In November last year we in Bristol were visited by Professor Massé, Professor of Surgery in Bordeaux. He gave us lectures on the aims and organization of the French blood-transfusion service, of which he is the regional head, and on the preservation of arterial grafts. Although his visit here was brief, we were able to show Professor Massé something of our hospitals, university and city, and the regional transfusion service.

THE CITY OF BORDEAUX

Bordeaux is a city of almost the same size as Bristol, lying at a bend in the broad river Garonne some sixty miles from its entry into the Atlantic. It has been the natural capital of its region, Aquitaine, for many centuries, and the remains of a Roman amphitheatre within the city testify to its importance in Roman days. Our hosts reminded us with pride that Bordeaux was an English city for three hundred years, beginning in 1154 when it became an English possession on the marriage of Henry II with Eleanor of Aquitaine. The Bordelais of those days evidently flourished under this arrangement, appreciating their independence from the throne of France as well as their convenient distance from the throne of England. Trade grew between Bordeaux and England, largely through the port of Bristol, and the main commodity shipped was wine, for which Bordeaux was already famous. Both the cathedral and the university were founded in the days of English rule, and later Bordeaux became the centre of the court of the Black Prince; it was here that his son, subsequently Richard II, was born. The English were finally defeated in 1453, in the Hundred Years' War, after which Bordeaux became a French city. The city's greatest days were, perhaps, in the eighteenth century, and there are many fine boulevards, squares and buildings, notably the magnificent theatre, dating from this period. The main industrial activity of Bordeaux and its countryside is still the wine trade, now recovering from the effects of the war; we spent some pleasant and instructive hours in the famous wine-producing districts near Bordeaux—Médoc, Graves and Sauternes—seeing and tasting their products. We quickly realized, too, that Bordeaux has a well-deserved reputation for food as well as for wine. In touring the city we were interested to see the big new stadium and sports park, where the influence of Spain and the Basque country is shown by the occasional holding of a bull-fight or pelota match.

HOSPITALS AND MEDICAL SERVICES

We visited a number of hospitals in and near Bordeaux. For the most part they are housed in old and undistinguished buildings, in which the processes of expansion and modernization are as painful and costly as they are in England. However, reconstruction is going ahead, if slowly. On the whole, laboratory equipment seemed at least

equal to ours, although wards in the older buildings were often dreary. There was certainly no lack of talent, industry and ingenuity among the medical staff. My own particular interest was in the cardiac department, under the charge of Dr. Broustet. This was evidently not a show place, and was presented rather apologetically; nevertheless space was ample, laboratory equipment was in many ways better than our own, and I was impressed by the work on cardiac and pulmonary function being carried out by Dr. Bricaud. On one of my visits I watched a mitral valvotomy performed by Dr. Dubourg, and in the course of discussion found that they had done this operation nearly as often as we have in Bristol, and that our experiences with it are very similar. The chest hospital and sanatorium newly built a few miles from Bordeaux was magnificent, and lavishly equipped. Here again the talent and character of the medical staff were impressive, and I was interested to see a colour film of bronchoscopic appearances in various conditions made by Dr. Fréour.

Medical care in France is based upon a system of compulsory insurance which does not go as far as ours in England. The patient receives attention from his doctor, from a specialist of his own choice, or in hospital, and the cost is defrayed to a limited extent by the insurance. Those who can, prefer to enter nursing homes or private clinics, where the fees, of course, may be far above the sum provided by the insurance. Nearly all doctors rely upon private practice for their income, for the remuneration from insurance and hospital work is low, and the cost of living is high. Consequently the general practitioner does not have the standing that he does in England, particularly in the French cities. The majority of doctors obviously prefer to specialize when they can, and not uncommonly help to establish clinics or hospitals which are based upon private investment, or a mixture of private and communal investment.

We found on all sides a most lively interest in the National Health Service, tempered by a polite scepticism. Vigorous discussions ensued on the relationships between the universities and the Health Service: strong views were held for and against the desirability of whole-time posts for medical staff in teaching hospitals. Conservative French opinion is deeply attached to the ideals of independence and private enterprise, while the younger generation, being less committed to the *clientèle privé* as the basis of existence, is more prepared to accept some form of partly-salaried health service, or whole-time posts in certain hospitals, However, conthe main flict of opinion seems yet to come.

THE MEDICAL FACULTY

The medical school at Bordeaux is not only several hundred years older than ours, but is also very much bigger. This is mainly because French medical schools and universities, which are financed and controlled by the Ministry of Education, are bound to accept all students capable of entering, without limitation of numbers. Students fees are a fraction of ours, and are virtually only token payments. The annual entry of students averages about 300, compared with our 60, resulting in phenomenal difficulties in teaching, particularly in laboratory work and clinical training. The medical course lasts six years. After spending the first year on science subjects, students enter the Faculty of Medicine, and throughout the second to sixth years it is the custom to spend mornings at the hospitals and afternoons in the faculty at lectures or in the laboratory. There are annual examinations, the main clinical examinations coming after the fifth year, and the fail rate in the earlier years may be as high as 20–30 per cent. If all goes well, however, the student submits a thesis after his sixth year, and acceptance of this brings him the degree of M.D. and the right to practise.

For the rank and file of students, clinical training and practical experience must necessarily be very limited. However, after the second, third and fourth years students compete by examination for some fifty positions each year as "externs". Externs work as clinical assistants in various departments in rotation in the mornings, continuing their studies in the afternoons; they are non-resident, and receive a small emolument. After the fifth year ten "interns" are chosen each year by examination, and they

correspond to our resident house physicians and surgeons, and are paid some £50 per year. Some may serve a second or third year's internship, ultimately presenting their thesis for the degree of M.D. in the usual way. Subsequently these men will have the right to announce on their brass plates that they are "anciens interns des hôpitaux de Bordeaux".

A man wishing to go further in the teaching hospital will then compete by examination for the post of *chef de clinique* becoming a junior member of the university staff (and thus under the Ministry of Education) corresponding to our registrars and junior lecturers. A position on the staff of a non-teaching hospital, under the Ministry of Health, may be gained, again by competition as *médecin des hôpitaux* or *chirurgien des hôpitaux*. Further advancement in the teaching hospital means a final examination for the position of *professeur agrégé* at about the age of 40. Most, but not all, *professeurs agrégés* can hope to be elected to a full professorship some ten years later. None of these posts carry more than a nominal salary, so that every doctor above the level of intern has to take up private practice for a living, usually in a specialty. Higher, or specialist, degrees are uncommon in France, and the energies of ambitious doctors are evidently fully absorbed by the system of advancement by competitive examination.

THE NAVAL MEDICAL SCHOOL

At Bordeaux the annual entry of 300 students includes about 100 from the French naval medical school. This is the only one of its kind in France. and has its parallel in the army medical school in Lyons. A young man wishing to serve as a doctor in the French navy or oversea possessions may enrol in the navy after passing his *baccalaureat* at the age of 17 or 18. He then lives in the naval medical college in Bordeaux under a modified naval discipline, and is uniformed as a cadet. He is there given his initial naval training and his introductory science course, and, if he wishes, such interesting extras as parachute jumping. He then enters the Bordeaux medical school with the second year and follows the usual course through with them. After graduating he is, of course, contracted to serve for a number of years in the navy or oversea. A keen interest is taken in rowing (*canotage*) at the naval medical school, and it is their crews who have competed with Bristol University crews in the past.

CONCLUSION

While in Bordeaux I was assured by M. Sigalas, the dean of the medical faculty, that a warm welcome would be given to any of our staff or students visiting the hospitals or medical school there, and on his visit to Bristol Professor Massé endorsed this invitation. The exchange of visitors between Bristol and Bordeaux continues, and it is clear that in other spheres as well as in medicine, friendship and understanding between Britain and France will be strengthened by this link between our cities.

My thanks are due in particular to Dr. Broustet and to M. Loiseau, Professor of English, for their great kindness, and the trouble that they took to make our visit so enjoyable.

SARCOIDOSIS TREATED WITH CORTISONE

Clinical Pathological Conference held at Canynge Hall on 5th February 1957

CHAIRMAN: PROFESSOR T. F. HEWER

Dr. J. M. Naish: This is a man of 37 who, at the age of 9, developed epilepsy. He had regular epileptic attacks between the ages of 9 and 23. I am not sure precisely what treatment he had during that time but after the attacks stopped at the age of 23 he went on having 1 gr. phenobarbitone at night and also a bromide mixture.

He first came under close medical scrutiny in the early part of 1954 when he was seen at the Central Health Clinic complaining of cough, loss of weight, scanty amount of sputum and some fever.

An X-ray of his chest showed a high right dome of the diaphragm and a rather large, glandular shadow in the right hilum. The E.S.R. was normal. There was no fever. The conclusion reached was that he was not suffering from tuberculosis but from some form of fibrosis of the lungs associated with bronchitis. Penicillin was recommended for treatment at home.

The next incident in this man's life was in September 1954, approximately nine months after the above X-ray was taken, when he was noticed to have rather a large "tummy" and complained of some abdominal pain. The following month he had rather more severe abdominal pain. His doctor sent him to Southmead Hospital to see Mr. Pocock, and he was investigated. His liver was enlarged and also his spleen. A barium meal showed no definite evidence of peptic ulcer, and liver function tests which were done at that time, in October 1954, showed the plasma globulin to be very much raised, being 5.5 g. per cent while the albumen was 3 g. per cent., E.S.R. 13 mm./hr. I think no definite conclusion was reached at this time as to the cause of the hepatosplenomegaly and he was discharged from hospital, the pain having settled down.

He was readmitted in January 1955, two and a half months later, with acute abdominal pain. An anterior perforated gastric ulcer was found at laparotomy. It was sewn up and he made quite a good recovery. He was discharged from Southmead to the recovery ward for surgical cases at Snowdon Road Hospital and while he was there his general condition was rather poor and he was about 2 stone under optimum weight so they asked me to see him. I was very interested by this combination of fibrosis in the lungs with enlarged liver and spleen, so we took him over to Southmead and did a liver biopsy with a "Terry" needle. This showed the typical appearances of Boeck's sarcoidosis, which was the diagnosis clinically suspected because of the rather peculiar combination of lung fibrosis, splenomegaly and hepatomegaly. The patient was reasonably well in himself. There was no fever and no change in peripheral blood count. Hb was about 85 per cent.

Acting on the assumption that sarcoidosis may be a reaction to tuberculous infection with massive production of fibrous tissue, this patient was treated with streptomycin, P.A.S. and cortisone. That treatment is followed, I should say, by about half the physicians in this country for patients with sarcoidosis who have substantial symptoms. We felt that with this man, who was underweight with a grossly enlarged liver and moderately enlarged spleen, it was justified. We kept him on this treatment for nearly a year, and watched him in Out-Patients.

In March 1955, seven months after the start of treatment, he had rather nasty paronychiae of his toes, oozing thin pus, so I decided to wean him off cortisone in view of the probability that cortisone was interfering with his response to infection.

We then readmitted him in January 1956. By this time he was off his cortisone and we consulted the surgeons who removed several of his toe-nails and the infection

cleared up. When all had healed we put him back on streptomycin, P.A.S., I.N.H. and cortisone 100 mg. per day. During the subsequent half-year, to July 1956, he improved very much, gained nearly 2 stone in weight and his liver and spleen decreased very much in size, the liver being barely felt. Cortisone was reduced to 75 mg. per day in May 1956 and to 50 mg. in October 1956.

During the whole of this time he was having a small dose of phenobarbitone because of this old history of epilepsy. He got back to work—he was a grocery manager—and he worked during the whole of the latter part of 1956. He had no trouble with epilepsy during the whole of this time. Then, on 21st November 1956, he started, quite suddenly, having numerous epileptic attacks. I am not sure of the precise treatment given by his own doctor but he was sent to hospital the following day and found to be dead on arrival—he died in the ambulance.

Here is a man, epileptic in childhood and early youth, who developed Boeck's sarcoidosis at the age of 36. This was treated with anti-tuberculous drugs and cortisone, which had good general effect with reduction in the size of liver and spleen. He died in status epilepticus after having had no epilepsy for fifteen years.

Professor C. Bruce Perry: Was there any change in the radiological picture of the chest over the eighteen months or so?

Dr. Naish: I think there was some clearance there.

Professor Perry: Did you think the shadow to the right of the heart was due to enlarged glands?

Dr. Naish: I thought there were some enlarged glands but also some partial collapse of a basal segment of the lower lobe.

Professor Perry: Is it true that he was given hydantoin for his epilepsy? It says so on the sheet.

Dr. Naish: I don't remember that he was and I don't know where that statement came from.

Dr. N. J. Brown: It says so in the case notes.

Professor Perry: So he did have fits which were treated?

Dr. Brown: He may have been put on it to prevent fits. It states in the notes that he was treated with it.

Dr. Naish: It certainly wasn't prescribed by us! We were never consulted about the epilepsy—he had not had any fits in all these fifteen years.

Dr. Brown: It may be that he had been put on it as a prophylactic measure before he came in and it was continued in hospital.

Professor Hewer: Dr. Mather has had a lot of experience in, and has made a special study of sarcoid. Perhaps he would like to comment on this case.

Dr. H. G. Mather: Yes. I think it is very unusual in as much as he had an enlarged liver as well as enlarged spleen. An enlarged liver in this disease is unusual. In a series of 93 cases no liver enlargement was demonstrated, although 14 per cent. had enlarged spleens. It has now become generally realized that many different manifestations of this condition are, in fact, all one disease. They used to be given various different names according to whether they were skin lesions or whether they were in the eye—uveitis—or whether they were in the bones. We now know that it is all one disease—sarcoidosis. A very important manifestation is uveitis and this is one of the reasons for making a firm diagnosis because it can be relieved by cortisone. We now know that the various skin manifestations are comparatively rare. Bone manifestations are even rarer and merely form part of the overall disease. We don't know anything about the cause. With regard to treatment, I entirely agree with Dr. Naish—we should give cortisone.

Professor Hewer: You say uveitis can be cured by cortisone?

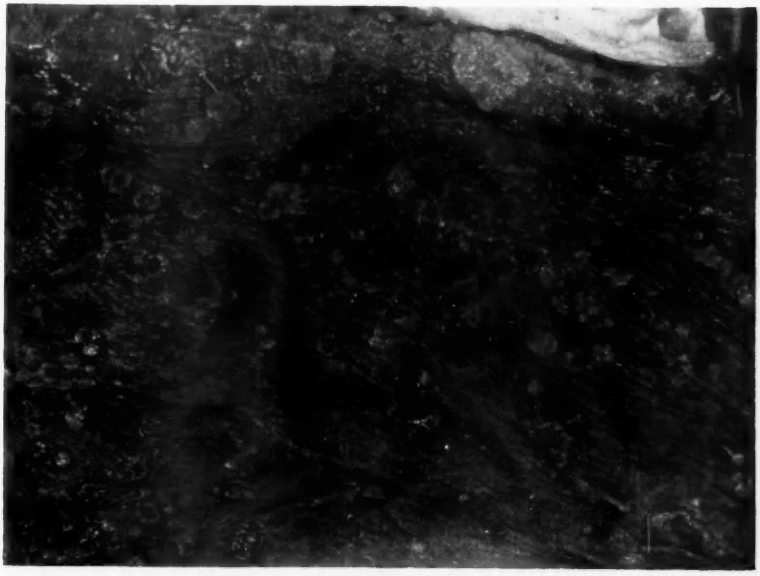
Dr. Mather: Yes. I think everyone with severe uveitis should have cortisone. I have seen several people go blind and this can be prevented by cortisone. This patient had no changes in his eyes.

Question: Is it a killing disease?

PLATE IX

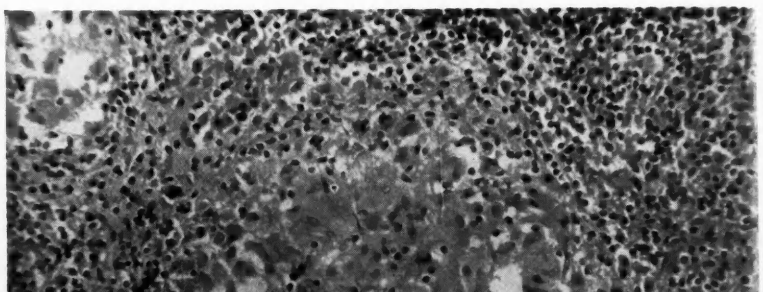
Spleen, showing the very numerous sarcoid nodules.

PLATE X



Surface of lung showing sub-pleural sarcoid lesions (× 1½).

PLATE XI



Photomicrograph of spleen (× 140) showing typical sarcoid lesion composed of epithelioid cells and multinucleated giant cells.

Dr. Mather: Very rarely. Many cases are asymptomatic and the bulk of people suffering from sarcoidosis are far best left alone; 63 per cent clear within a year without any treatment. Leave well alone unless there are symptoms and the most important symptom is uveitis.

Professor Hewer: Unless uveitis or other symptoms occur, you would prefer to do nothing?

Dr. Mather: Yes. It is a killing disease in only a small percentage of cases. We aim to prevent progressive fibrosis in the lungs.

Professor Perry: The tragedy is once fibrosis starts, cortisone does not help.

Dr. Mather: Quite true.

Professor Perry: Did any of your cases present with erythema nodosum?

Dr. Mather: Yes; 12 per cent.

Dr. N. J. Brown (*Presenting the autopsy findings*)*:* There is no mystery about this case. The diagnosis has already been given you and it is not going to be changed. The importance of this case is that it gives us an opportunity to study the lesions of Boeck's sarcoid at a stage when you don't usually see them. They don't die of it in this stage and if they die of sarcoid you only see " burnt out " lesions.

This man died of asphyxia due to status epilepticus, as a result of continued spasm of the muscles of respiration and possibly also of the larynx. By dying in this manner he afforded us a most rare opportunity of studying the lesions of another disease—sarcoid—from which he happened also to be suffering.

First of all a few words about the disease. It was first described as a skin disease by Jonathan Hutchison in 1869. It was first called sarcoid by Boeck in 1899 and he also described the histological appearances. Boeck was a Norwegian physician who died in 1917. The disease is an inflammatory one in which one finds granulomatous lesions of a characteristic type in various parts of the body. They all used to have different names; if they were in the skin, they were called Boeck's sarcoid; if in the eye—Heerfordt's disease or uveo-parotid fever; if in the bones—osteitis tuberculosa multiplex cystica, and so on. It is now realized that these different manifestations are all part of the same disease—sarcoidosis. Generalized sarcoidosis, as in this case, is sometimes called Schaumann's disease. Schaumann was a Swedish dermatologist who described the generalized type in 1914. You will notice the number of Scandinavian names associated with this disease; this is a reflection of the fact that the disease is commoner in Scandinavia than anywhere else in the world.

This case is a beautiful example of generalized sarcoidosis with lesions in many parts of the body. I shall show you a large number of pictures and describe the lesions to you as we go along.

Dr. Brown then showed lantern slides and photomicrographs of the various organs.

In the spleen (Plate IX), there were large numbers of whitish, nodular lesions each 1–2 cm. in diameter, spread evenly through the substance of the organ and appearing beneath the capsule. The spleen was greatly enlarged, being approximately six times the normal size.

In the liver, there were numerous similar lesions but the individual nodules were rather smaller.

There were enlarged lymph nodes all over the body, the largest one being approximately 2 cm. in diameter in the para-aortic region. For the most part they were discrete but in places they were matted together. Some showed a uniformly pink cut surface but in some there were pale miliary nodules.

The lungs showed whitish-pink, nodular lesions beneath the pleura (Plate X) but these were not easily seen on the cut surface although it was possible to palpate large numbers of nodular lesions throughout the substance of both lungs.

Microscopically, the lesions all had a similar appearance. The nodules were made up of aggregations of sarcoid follicles composed of large multinucleated giant cells, some epithelioid cells with a rather reticulated cytoplasm, and a surrounding zone of lymphocytic infiltration (Plate XI). The follicles had a clearly defined outline and

showed no caseation. No acid-fast bacilli were demonstrated in them. Many of the giant cells were particularly large and striking, one, in the lung, having as many as 57 nuclei. There was little surrounding fibrosis. The lesions differ from miliary tubercles in that there is no caseation, no acid-fast bacilli are seen, the giant cells are larger and the lesions are more discrete. Reticulin impregnation showed a fine reticulin pattern throughout the lesions, while in tuberculosis the reticulin pattern is usually destroyed. In the lung, sarcoid follicles were present in the walls of the bronchioles and were particularly frequent in the walls of small arteries, in some of which they infiltrated into the lumen with resulting thrombosis. In the lymph-nodes there were large masses of homogeneous eosinophilic material. This superficially resembles amyloid but does not take the characteristic pink stain with gentian violet. It is known as paramyloid and is a characteristic finding in sarcoidosis. A few lesions were demonstrated microscopically in the kidney but the largest number were found in liver, spleen, lung and lymph nodes. No sarcoid lesions were demonstrated in the brain or meninges.

The lesions were very typical of generalized sarcoidosis. Sometimes, in the giant cells, you see peculiar inclusions known as asteroid bodies. These were not easy to find in this case and unfortunately photographs were not available.

In conclusion, one might say a little about the theories of aetiology of this condition. Sarcoidosis has been variously considered to be:

(1) A chronic granuloma resembling tuberculosis but caused by some as yet undiscovered agent;
(2) An atypical form of tuberculosis;
(3) An atypical response on the part of the reticulo-endothelial system to tuberculosis or a form of hypersensitivity reaction.

Professor Hewer: One of the reasons for linking this disease in one's mind with tuberculosis is that the tuberculin reaction is usually firmly negative. Is that not so?

Dr. Mather: No. That is not true; 44 per cent. are negative to tuberculin 1 : 100 dilution. The diagnosis of sarcoidosis cannot be ruled out by a positive tuberculin test.

Dr. Naish: Did some of your cases convert from negative to positive?

Dr. Mather: No.

Dr. J. E. Cates: This is one of the most important cases we have had here for a long time. We have had a man on P.A.S., streptomycin and cortisone for a long time and he has accidentally died. I wonder whether you would now say that cortisone and other things have had any effect on him at all. This ought to help us decide whether such treatment will help us with our future cases.

Dr. Naish: I was merely following the example of authorities on the subject who had—at the time when this man first came under observation—noticed considerable improvement in the radiological appearance of the lung lesions after this treatment. And with this man there is no doubt that his liver decreased in size very considerably while under treatment but that may perfectly well have been the natural course of events. I would be the last to say treatment had any effect at all, but one believed it was honestly the best thing to do for the chap. I don't know whether one can argue an awful lot from the fact that his sarcoid lesions are still there.

Dr. Mather: I quite agree. One must not be afraid to treat sarcoid with potentially dangerous drugs. One treats many diseases without completely curing the lesions. We treat mitral stenosis, but there is still mitral stenosis at post-mortem. The treatment is to relieve symptoms. If they are got back to work it is a good job.

Dr. O. C. Lloyd: What happens if you treat sarcoidosis with cortisone without streptomycin and P.A.S. Do some develop tuberculosis if you give them that?

Dr. Mather: They may, but you may have been dealing with tubercle all along; hence it is important to make a firm diagnosis of sarcoidosis. People are afraid to give cortisone alone in case some die of tuberculosis, so we give P.A.S. and streptomycin as well as cortisone to prevent tuberculosis but many physicians now treat

sarcoid with cortisone alone without any serious complication. There is a funny association between tuberculin sensitivity and sarcoid because if you took 100 normal people, you would expect less than 44 per cent. negative to 1 : 100 dilution. It is very difficult to make people with sarcoid tuberculin positive even by giving B.C.G. Cases of Hodgkin's disease have a negative Mantoux in a similar proportion to patients with sarcoidosis, suggesting that the anergy may be due to the reticulo-endothelial involvement.

Notes and News

The Board of the South-West Faculty of the College of General Practitioners has decided to institute an annual lecture in memory of the late Dr. A. H. Gale. It will be given each year prior to the Annual Meeting of the Faculty. The inaugural lecture will be given at Torquay on Sunday, 13th October, by Dr. R. S. Hope-Simpson, of Cirencester, on "Opportunities and Pitfalls of Research in General Practice".

UNIVERSITY OF BRISTOL

M.D.

Sanerkin, N. G.

M.B., Ch.B.

WITH SECOND-CLASS HONOURS

Hockin, Janette A.
Shortman, S. C. J.

Pass

Akinsete, F. I.
Betteridge, T. J.
Bland, Pauline M.
Bowden, D. E.
Brewer, C. C. L.
Burland, W. L.
Counsell, G. F.
Daniels, S. M.
Dansie, Norma St. C.
Douglas, H. G. K.
Dowell, Bethia M. (*with Distinction in Public Health*).
English, J. M.
Faint, Sheila A.
Falkner, C. G.
Faulkner, D.
Fraser, I. D.
Fung, Joyce
Innes, D. M.

Curtis, G. T.
Millen, H. E.

Jenkins, C. W.
King, Audrey
Koney, J. O.
Manley, G. W.
Moffatt, Jean (*with Distinction in Public Health*).
Morgan, A.
Onubogu, H. O. N.
Pengelly, D. C.
Rackham, K. T.
Robertson, D. A. S.
Rose, A. L.
Shaw, J. F.
Sheard, M. E.
Stock, P. R.
Sutton, G. B.
Taylor, Margaret
Thompson, I
Weeks, D. C.
Wotherspoon, H. G.

Prescot, R. E.
Stamper, J. R.
Zutshi, D. W.

EXAMINATION RESULTS

M.R.C.P.: D. R. Coles.
L.R.C.P., M.R.C.S.: R. J. A. Brown.

PRIZES

Foyle Prize for 1957: G. Neale.
Edward Fawcett Memorial Prize for 1957: G. Neale.

Appointments

UNITED BRISTOL HOSPITALS

MARCH TO JUNE, 1957

Name	*Appointment*	*Formerly*
MACQUEEN, I. G., M.B., B.S., F.R.C.S.	Registrar in Orthopaedic Surgery.	Residential Surgical Officer, Tunbridge Wells Group.
EPSZTEYN, Miss L., M.B., CH.B., D.R.C.O.G.	Registrar in Obstetrics, and Gynaecology.	Registrar, Perth Royal Infirmary.
DOYLE, F. H., M.B., CH.B., D.M.R.D.	Senior Registrar i n Radiodiagnosis.	Registrar in U.B.H.
BURLEY, M. A., L.D.S., (EDIN.)	Registrar in Orthodontics.	Dental Officer, Colonial Service, Hong Kong.

Dr. H. M. Leather, M.B., CH.B., M.R.C.P., has resumed his appointment as Senior Registrar in Medicine on completion of his appointment as First Assistant in Medicine at Makerere College, Kampala, Uganda.

Dr. W. A. W. Dutton, M.B., M.R.C.O.G., has been appointed Tutor in Obstetrics and Gynaecology, Queen's University, Kingston, Ontario.

SOUTH WESTERN REGIONAL HOSPITAL BOARD

MAY TO JUNE, 1957

BRISTOL

Name	*Appointment*	*Formerly*
BENNETT, MRS. M. O. M.D., OBST., (LOND.), CH.M. (BRIST.), M.R.C.O.G., F.R.C.S. (ENG.).	Consultant Obstetrician and Gynaecologist, Southmead Hospital.	Clinical Assistant in Obstetrics, U.B.H.
CROSSLEY, JOHN, M.B., B.CHIR. (CANTAB.), F.R.C.S. (ENG.), M.R.C.O.G.	Consultant Obstetrician and Gynaecologist, Southmead Hospital.	Senior Registrar, St. Thomas's and Lambeth Hospitals.
WILLIS, AUDREY S., B.A., (CANTAB)., M.B., B.CHIR. M.R.C.P.	Senior Medical Registrar, Frenchay Hospital.	Registrar in Department of Experimental Medicine, Cambridge.
SOLLING, M. C. R., M.B., B.S. (SYDNEY).	Surgical Registrar, Cossham-Frenchay Group of Hospitals.	S.H.O. in General & Orthopaedic Sgy., North Middlesex Hospital.

NORTH GLOUCESTERSHIRE

Name	*Appointment*	*Formerly*
FERGUSON, J., M.B., B.S. (MELBOURNE).	Medical Registrar, Gloucestershire Royal Hospital.	Locum tenens appt. at St. Matthew's Hospital, London.
GREENWOOD, R. K., M.B., B.CHIR. (CANTAB.), F.R.C.S. (ENG.).	Surgical Registrar, Gloucestershire Royal Hospital.	Resident Surgical Officer, Royal Devon and Exeter Hospital.
HANNA, F. A., M.B., B.CH., B.A.O., D.M.R.T., (BRIST.).	Consultant Radiotherapist, North Gloucestershire Clinical Area.	S.H.M.O., North Gloucestershire Clinical Area.

BATH

Name	*Appointment*	*Formerly*
BAZELEY, R. W., M.B., B.S. (LOND.).	Re-appointed for further year as Clinical Assistant in General Medicine, Bath Group of Hospitals.	In general practice, Radstock.
ELLISON, D. J., M.B., CH.B., (EDIN.), M.R.C.P. (EDIN.).	Re-appointed for further year as Clinical Assistant in General Medicine, Bath Group of Hospitals.	In general practice, Melksham.
RONN, H. H., M.B., B.S. (LOND.), M.R.C.P. (LOND.).	Re-appointed for further year as Clinical Assistant in General Medicine, Bath Group of Hospitals.	In general practice, Westbury.
PINCHING, J., B.M., B.CH. (OXON.), M.R.C.P. (LOND.).	Appointed for one year as Clinical Assistant in General Medicine, Bath Group of Hospitals.	In general practice, Wells.
HIGGINS, P. J., M.B., B.CHIR., (CANTAB.).	Appointed for one year as Clinical Assistant in Paediatrics, Royal United Hospital, Bath.	In general practice, Westbury.
LUMB, G. N., M.B., B.S. (LOND.), F.R.C.S. (ENG.)	Surgical Registrar, Bath Group of Hospitals.	S.H.O. in Urology, Buchanan Hospital, St. Leonards-on-Sea.
PRINGLE, H. M., M.B., B.S. (SYDNEY).	Surgical Registrar, Bath Group of Hospitals.	Surgical Registrar, West Suffolk General Hospital.

DEVON AND CORNWALL

Name	*Appointment*	*Formerly*
TROTTER, J. K., M.R.C.S., L.R.C.P., D.A., F.F.A., R.C.S.	Consultant Anaesthetist, West Cornwall Clinical Area.	S.H.M.O., High Wycombe & Amersham Hospital Group.
ARTHUR, L. J. H., M.B., B.CHIR. (CAMB.), D. (OBST.) R.C.O.G., M.R.C.P. (LOND.)	Paediatric Registrar, South Devon and East Cornwall Hospital, Plymouth (joint appt. with the U.B.H.	Medical Registrar to the Professorial Unit, Royal Victoria Infirmary, Newcastle-upon-Tyne.
BARLOW, D. J. H., M.B., B.CH. (WITWATERSRAND).	Registrar in Psychiatry, Moorhaven Hospital, Ivybridge.	S.H.O., Fulbourn Hospital, near Cambridge.
DHILLON, B. S., M.B., B.S. (PUNJAB)., F.R.C.S. (EDIN. AND ENG.).	Senior Surgical Registrar to the Thoracic Unit, Hawkmoor Chest Hospital, Bovey Tracey.	Registrar to Neurosurgical Department, Sheffeld Royal Infirmary.